MEDICAL STAFF CONFERENCE

Medical Complications of Heroin Addiction

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. Sydney E. Salmon and Robert W. Schrier, Assistant Professors of Medicine, under the direction of Dr. Lloyd H. Smith, Jr., Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, San Francisco, Ca. 94122.

DR. SMITH:* We are pleased to have Dr. Charles Becker with us today to discuss the very important problem of heroin addiction.

DR. BECKER:† Ten months ago a 20-bed Detoxification Unit was opened at San Francisco General Hospital to aid patients who had acute and chronic alcohol problems and needed care in a hospital. Since that time approximately 700 patients have been admitted and some 200 consultations have been answered concerning many different drug abuse and clinical pharmacology problems. In attempting to be responsive to the community problem of alcoholism we learned that the public and private agencies which referred alcoholic patients to our unit also had a need to refer narcotics abusers. The scope of the Detoxification Unit was therefore broadened to admit patients needing management of complications of heroin abuse. This expansion has led to a broad learning experience, both challenging and frustrating, for the students, house staff and paramedical personnel. Today I would like to review for you our experience with these patients and to emphasize the magnitude of heroin abuse, the chemical and pharmacological features of heroin and the medical sequelae of heroin addiction.

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Vocabulary of the Addict

One of our first observations was that we would have to broaden our vocabulary to communicate with these patients. When asking for *Charlie* a patient in withdrawal period was not referring to a person but requesting cocaine. *Cotton* is the filter that is used in the spoon of the heated heroin while it is being drawn up into the needle. Some times this cotton is soaked and used as a marginal source of narcotic. The *spike* is the needle used to inject heroin. *Flash* or *rush* refers to the acute effects of the intravenous use of heroin which occur a few seconds after injection and are one of the prime goals of the narcotic user. *Monita* is a Spanish-Mexican slang word for the milk-sugar adulteration of heroin. *Bonita*, used in its special sense mainly in Harlem, is another term for the milk-sugar quinine adulterants. (The quinine is used to prevent the buyer from tasting top grade heroin, which has a bitter taste.) Intravenous quinine is associated with dilation of the blood vessels of the face and contributes to the flash. When we first heard *P.G.* we thought it meant Procter & Gamble, but we soon learned that it was argot for paregoric, which contains 0.421 percent of opium. When the paregoric is heated the camphor alcohol is evaporated, leaving the opiate. The intravenous use of paregoric is associated with sclerosing of

veins. It has been pointed out that people with injection scars in the neck most often are users of paregoric, the antecubital veins having been sclerosed by the use of P.G. *Hype* is the entire paraphernalia that is needed to inject heroin. *Speedball* refers to a combination injection of an *upper* (cocaine) and a *downer* (heroin). *Blues* are amobarbital (amytal®) or pyribenzamine, *yellows* are pentobarbital (Nembutal®) also called *yellow jackets,* and *reds* are secobarbital (Seconal®) and *whites* are benzedrine. *Rainbows* is a term for an assorted color of barbiturates of all varieties, and *crystals* are methedrine. Some knowledge of this argot is necessary to communicate with the heroin addict.

Incidence of Heroin Addiction

How prevalent is this problem of heroin addiction? One of us here could conclude that it was endemic, while another might think it rare, depending upon our rotations within the University hospital medical residency program. For example, in survey of a 12-month period of admissions to Moffitt Hospital we found a total of four patients who had a diagnosis of heroin abuse, in one of them heroin addiction as the primary diagnosis, and the other three had been transferred from the San Francisco General Hospital to Moffitt Hospital for heart surgery. In a comparable period, with a comparable number of admissions, there were several hundred patients admitted to the San Francisco General Hospital for heroin-related problems. A review of newspaper statistics reveals a range in the reported number of heroin addicts in the United States from 125,000 to 250,000. In San Francisco it is estimated there are between 5,000 and 10,000 heroin abusers. One of the most distressing aspects of the heroin problem is that a minimum of 10 to 15 percent of American servicemen are using high-grade, inexpensive heroin in Vietnam. These servicemen are then returning to the United States and finding the heroin quality is inferior and the cost much greater to maintain a comparable habit.

A recent review by Fort[1] suggests that 1 percent of college students have tried heroin and that in a Berkeley community 5 to 8 percent of the high school seniors had tried heroin.[1] A recent editorial in *Science* pointed out that drug abuse, primarily with narcotics, is the leading cause of death in New York City in the age group 15 to 35 years. The same editorial pointed out that there were at least 900 deaths a year related to heroin abuse.[2] In the city of San Francisco there were 56 reported deaths from heroin overdose last year, according to the coroner's office. One explanation for this high death rate may be the wide range of drug strength. The Office of the Medical Examiner in New York City recently analyzed 132 street samples of all drugs purported to contain heroin and found that 12 contained no heroin at all and among the remainder the concentration ranged from less than 1 percent to 77 percent. In California the average concentration of street heroin is approximately 2 percent.

Physicians are at special risk of narcotic abuse. Many articles have emphasized that between 1 out of 40 to 1 out of 100 physicians use hard narcotics in a regular way.[3]

Senator Hughes of Iowa, who has been a strong proponent of legislation to deal with drug abuse and alcohol problems, recently estimated that there is a 10 billion dollars per year economic loss from the use of narcotics. St. Luke's Hospital in New York recently compared the cost of maintaining 81 addicts on and off methadone therapy and estimated a net saving to society of more than $800,000 when the addicts were maintained on methadone. In spite of the magnitude of the problem of heroin abuse, there are only ten to twenty thousand patients in any treatment program.

Source of Heroin

What is heroin and where does it come from? The exact date when the mind-altering effects of the use of the extracts of the opium poppy plant were discovered is not known. The discovery is often attributed to the Egyptians, who used tincture of opium to prevent excessive crying in children. Greeks and Romans were aware of the mind altering effects of some of the parts of the poppy plant. Opium was carried to Persia and China in the 9th Century by Arab traders and soon achieved wide popularity. In 1803 a young German pharmacist isolated and described an opium alkaloid that he named morphine, after Morpheus, the Greek god of dreams. Between 1834 and 1858 two so-called "opium wars" were fought and won by the British when Chinese officials attempted to stop trade and seize many of the opium exports. With the invention of the

hypodermic needle in 1840 began the much more severe variety of compulsive narcotic drug use which became rampant during the Civil War in the United States. In an attempt to treat medically the growing narcotic addiction problem, acetylation of the morphine molecule was successfully performed in 1898, leading to the "magic cure" of opium addiction, namely heroin. It should be emphasized that for years heroin was considered a safe, nonaddicting substitute for morphine. Gradually the tremendous mind-altering effects of the intravenous use of heroin became known. International travel by young Americans during World War I, World War II, the Korean and Vietnam wars exposed more and more individuals to these effects of heroin. Currently most opium is obtained from Turkey, Burma and Thailand, shipped to illicit laboratories in Europe where the morphine base is converted to heroin, which is shipped to the United States.

Heroin is a very light, white, bitter chemical which may be adulterated or combined with lactose-mannitol and quinine to mask its bitter taste. Brown heroin is characteristically derived from Mexican sources. In Turkey 20 pounds of raw opium will bring a farmer approximately $300, and by the time the final product reaches the United States it is worth $250,000. Heroin is often injected subcutaneously ("skin-popped") or applied to the nasal mucosa ("snorted") before intravenous or "mainline" therapy is attempted.

Since tolerance to heroin develops rapidly, the cost of effective amounts soon becomes unbearable and the patient's life-style centers around obtaining enough money to buy what he needs. To support such a growing habit the heroin addict must classically work in any or several of four occupations—"deal, steal, pimp or whore." It should be emphasized that patients who seek help in the hospital for detoxification or for medical sequelae of the use of narcotics are really addict failures: They are sick because they have failed to obtain the narcotics. Most heroin addicts are "well" and "hustling" to support their habit.

Definition of Addiction

No word has caused more confusion and misunderstanding than *addiction.* To most pharmacologists, addiction has three components, namely a behavior component referring to psychological craving for a drug; a second component involving a physical dependence on the drug with withdrawal symptoms occurring when the medicine is discontinued; and a third component of tolerance whereby progressively more drug is required to achieve the same effect.

In recent years it has become clear that some drugs, commonly referred to as addicting, are not actually addicting according to the above definition. These drugs are now referred to as entailing "psychological dependence," more commonly known as habituation. Habituation refers to an individual's becoming accustomed to an agent through regular usage so that when the drug is no longer available he becomes restless, irritable or ill at ease. Certainly all of the mind-altering drugs can with regular use lead to psychological dependence, but whether dependence on the drug is significantly impairing the user's life function and whether withdrawal symptoms and tolerance have developed needs further evaluation in each patient. Because of the controversy in definition, most physicians in the field have preferred to refer to heroin addicts as "compulsive narcotic users." Regardless of definition, heroin addiction involves a rapid tolerance to the drug. The explanation for the dramatic increase in dose requirements, which may approach 100-fold, is not well understood but is a pharmacological characteristic of many narcotic drugs.

Chemical Features of Heroin

The narcotic alkaloids occur in the sticky brown gum that is scraped from the surface of an unripe seed pod of the poppy plant. Generally the raw alkaloids are placed in two chemical categories: phenanthine derivatives which characterize morphine and codeine, and the benzylisoquinolone alkaloids representing papaverine-like drugs. Heroin, diacetylmorphine, has substituted acetyl groups in the R_1 and R_2 positions of the phenanthrene nucleus with an unsaturated double bond in positions 7 and 8, and hydrogens in positions 3 and 4. The chemical structure of the opium alkaloids is often written to emphasize the common structure involving a tertiary nitrogen, a short hydrocarbon chain, and a flat aryl group. Pharmacology texts classically describe a hypothetical receptor surface which can accept the narcotic agent but not a mirror image. Narcotic antagonists can also attach to the receptor and reduce the potency of the agonists.[4]

Heroin is rapidly hydrolyzed to monoacetyl-

morphine (MAM), which in turn is hydrolyzed to morphine. In adult patients the blood-brain barrier tends to impede the entry of morphine into the brain. Because it is more lipid-soluble than morphine, this barrier is considerably less effective against heroin, and heroin therefore gives a better "rush-flash" and more rapid action. Most current evidence now suggests that morphine is responsible for the pharmacological actions of heroin. Heroin is primarily excreted in the urine, largely as free and conjugated morphine, and 10 percent is excreted in the bile. Heroin is approximately 2 to 4 times as potent as morphine in relieving pain.[4]

Pharmacological Effects of Heroin

The pharmacological effects of heroin are striking. Within seconds after the intravenous injection of the medication there are dramatic effects —sensations in the upper abdominal region caused by pylorospasm, deepening of the voice, and central nervous system depression associated with nodding, euphoria and drowsiness. With larger doses, there is suppression of respiration, heart rate and blood pressure. It is important to emphasize that the hypotensive effects of narcotics in general are enhanced by the phenothiazines. We have been impressed with the frequency with which heroin addicts are given phenothiazines by unsuspecting physicians and then have pronounced hypotensive effects from intravenous heroin. The patients may become hypothermic and spinal fluid pressure will increase. Histamine is released, leading to urticaria or unusual cutaneous sensations. This may be very disturbing to some addicts and often varies with the purity of the preparation. The cough reflex is suppressed and if vomiting occurs the upper airway may be unprotected and lead to aspiration pneumonia. From a clinical point of view, tolerance may develop to many of the above pharmacological effects of heroin, but fortunately little tolerance develops to the pupillary constriction and the overall gastrointestinal response of constipation. The contraction of the pupils in a comatose patient is a key physical finding in diagnosing acute narcotic overdoses.

Diagnosis and Treatment of Acute Heroin Overdose

Acute heroin overdose is frequently seen in the Mission Emergency Room at the San Francisco General Hospital when new supplies of more potent heroin become available in the streets. It is also very common that the patient who is admitted to hospital for concomitant medical problems or detoxification has withdrawal symptoms and then returns to his previous habit with a dramatically decreased tolerance. Then if immediately on release he uses the same amount he was using before, he may become acutely comatose with slow, shallow, irregular respirations, a slow pulse, decreased blood pressure and small pupils. The key is to observe the patient for needle marks, including careful observation of the genitalia, the tongue and between toes as important occult portals of entry. We have observed at least ten patients who have been using narcotics in the hospital by injecting the drug into intravenous lines. A patient who becomes comatose and has contraction of pupils while in the hospital may have received illicit narcotics. The treatment of the acute intoxication depends upon expert respiratory support and the judicious use of narcotic antagonists. These antagonists themselves can exert narcotic action if given in excess quantities. The relation of agonist and antagonist is usually described as being competitive, and the relationship is generally viewed as displacement of the agonist from the receptor sight. Nalorphine (Nalline®) is administered in a dose of 2.5 to 5 mg and is usually dispensed in 1 and 2 ml ampules of 5 mg per ml. Levallorphan tartrate (Lorfan®) is administered in a dose of 0.5 to 1.0 mg and comes in 1 ml and 10 ml vials of 1 mg per ml. An abrupt withdrawal syndrome can be precipitated by large doses of antagonists. Blood gases, pupil size and withdrawal symptoms must be monitored in determining correct dosing with narcotic antagonists.

Heroin Withdrawal

The onset of withdrawal symptoms after the last dose of heroin varies with the dose and magnitude of the habit. The symptoms of the heroin withdrawal syndrome include anxiety, yawning, lacrimation, rhinorrhea, profuse sweating, dilation of the pupils, vomiting, muscle aches and hot-and-cold flashes. Hypertension, hyperthermia and increased respiration are common. In contrast to withdrawal from sedative drugs such as alcohol and barbiturates, seizure and headache are uncommon. The duration of withdrawal symptoms is widely variable and is greatly altered by medication used in treatment. We have had a most

difficult time in gauging the narcotic requirements for patients in heroin withdrawal. We have found that the only effective way to identify the degree of withdrawal is to give a placebo in an orange flavored drink (Tang) with quinine and quinidine added to simulate the bitterness of methadone. Empirically, with house officers testing, we are able to detect a minimum of 5 mg of methadone in Tang. House staff and patients were not able to detect a difference in taste between methadone and quinidine at any dose.

By adopting a principle of testing all patients initially with a placebo, we have dramatically decreased the symptoms of withdrawal in some patients without giving any narcotics. It is generally felt that the half-life of methadone in heroin addicts is in the neighborhood of 36 hours. During severe withdrawal periods the clinical half-life may be much shorter, but in general patients are treated only once daily with methadone an initial average dose of 20 mg. Often patients will overestimate their habit in an attempt to obtain more narcotics from the physician. If tolerance to heroin is not present, giving methadone can cause severe overdose reactions. With regard to treatment for a methadone overdose, it must be emphasized that any of the narcotic antagonists must be given for a longer period in repeated doses than with heroin overdose since the half-life of methadone is longer than heroin's.

We have observed that many patients with the most severe withdrawal are very febrile and have complicating medical problems which must be searched for and treated appropriately. In our experience, infection is the most common precipitating event. Antibiotics must be reserved for the treatment of a specific infectious agent; prophylactic coverage of all heroin addicts in withdrawal would seem inappropriate and potentially dangerous. When possible, physicians should attempt to avoid intravenous infusions in these patients since this provides a ready means for illicit drug use. We allow no visitors during the entire withdrawal period, for we have sometimes seen guests administer heroin to our patients. Most of all, we should emphasize that methadone withdrawal alone is a uniformly unsuccessful way of treating the long-term problem of heroin addiction in that the vast majority of addicts who use methadone withdrawal in the hospital will return to the addiction unless the appropriate rehabilitative programs are established.

TABLE 1.—*Medical Complications of Heroin Addiction*

Increased mortality
Pulmonary complications
Hepatitis
Tetanus
Malaria
Bacterial endocarditis
Infection
Perplexing Serologic
? Necrotizing Angiitis (Citron)

Medical Complications (Table 1)

It is not surprising that the self-administration of adulterated opiates of variable concentration in large doses in an unsterile manner is frequently associated with severe medical complications. Although heroin addiction is commonly considered to be primarily a social and psychiatric problem, several interesting medical complications have become increasingly evident. However, the vast majority of patients with long-term and compulsive use of narcotics seem remarkably free of severe medical complications.

There is substantial evidence that the mortality rates for addicts are considerably higher than for age-matched controls. A definite increase in mortality rates in the United States has been described: as 16 per 1000 for addicts under 30 years of age, and 31 per 1000 for those over 30 years of age.* Similar reports have come from other countries. In New York City, where careful records are kept of the total number of deaths from narcotic addiction, the absolute number of deaths has climbed steadily over the past few years. There were approximately 1000 deaths from narcotic overdose in New York City last year, and approximately 60 in San Francisco.[5] The most feared complication of narcotic abuse, overdose, kills an estimated 1 percent of addicts in New York each year. The vast majority of addicts experience overdose at least once during the course of their drug abuse. It is clear that after the addict has been incarcerated in a hospital or jail, or otherwise kept from drug use, he returns to his previous drug habit and often disregards his abstinence-related loss of tolerance. He then may greatly overdose himself. The neophite narcotic addict, in an attempt to imitate more experienced users, may use inordinately large amounts of nar-

*Normal Death Rate is Age 15-29, 1.2/1000 in 1970
Age 30-59, 9.6/1000 in 1970
Source: San Francisco Department of Public Health

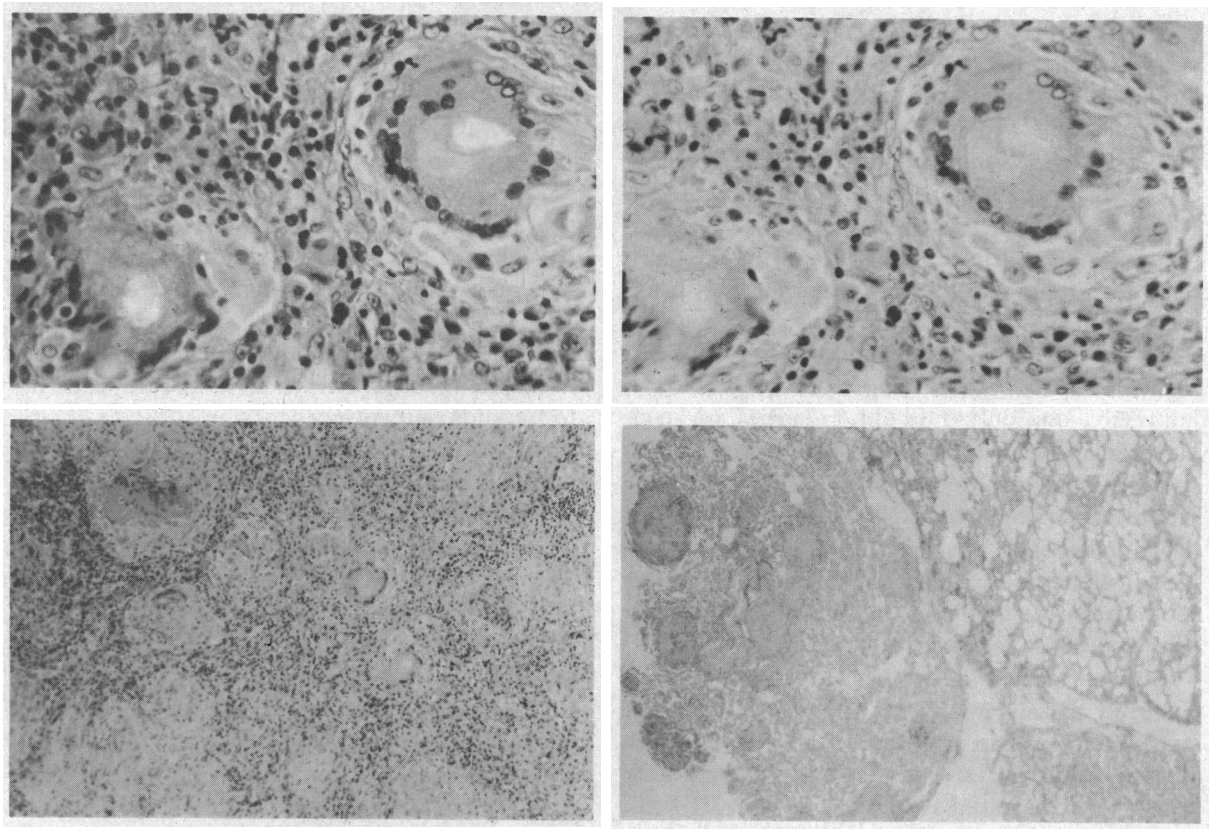

Figure 1.—Lung biopsy sections illustrating non-caseating talc granulomas with large giant cells and polarizing granules.

cotics. It is also clear that narcotic distributors, when expecting arrest or attempting to remove police informants, may pass large amounts of virtually pure heroin to unsuspecting users.

Since the addict injects himself intravenously with adulterated narcotics he may introduce particles of cotton or other filters of heroin that embolize in the lungs. This may lead to granulomas or pulmonary fibrosis and may mimic a wide variety of pulmonary infiltrates including sarcoidosis. Patients who inject paregoric and pyribenzamine *(blue velvet)* are especially liable to pulmonary hypertension and talc granuloma.

The lung reactions associated with heroin abuse are varied. Figure 1 shows sections of lung biopsy material from a young drug user who was recently evaluated at San Francisco General Hospital for progressive dyspnea and pulmonary infiltrates. She was clinically suspected of having sarcoidosis. Biopsy showed granulomas which were periodic acid Schiff negative and polarized under the microscope. These findings are characteristic of talc granuloma.

Acute pulmonary edema secondary to opiates was first described by Osler in 1880. We have recently observed four patients with acute pulmonary edema secondary to heroin overdose. Since this pulmonary edema picture was described many years before the discovery of heroin and its subsequent adulteration, it is presumed that the narcotics alone may be responsible. A recent review of many chest films of heroin addicts pointed out that pulmonary edema may occur in many patients with heroin overdose.[6] The most common x-ray finding of this pulmonary edema is one of fluffy, ill-defined bilateral coalescent densities. Treatment with narcotic antagonists may abruptly improve the pulmonary changes. A recent paper suggested that the pathogenesis of heroin-induced pulmonary edema may be due to increased permeability of the pulmonary capillaries since some patients have normal pulmonary artery pressures and the extravasated edema fluid has a significantly greater proportion of total protein than is seen in patients with arteriosclerotic heart disease and pulmonary edema.[7,8]

Hepatitis is one of the most frequent side effects of the chronic abuse of narcotics. It has recently been estimated that 10 to 15 percent of addicts have pronounced biochemical abnormalities consistent with acute hepatitis. Another 60 percent have distinct though far less impressive biochemical abnormalities which are usually attributed to chronic hepatitis, perhaps modified in some way by continued use of the drug.[9,10,11] Attempts to reproduce similar abnormalities in experimental animals have been unsuccessful. Despite the abnormal liver function tests in heroin addicts, results of liver biopsy are generally unimpressive and only mild abnormalities are seen. Holmes and coworkers recently demonstrated striking hypertrophy of the smooth endoplasmic reticulum, as might be expected in persons using drugs metabolized by hepatic microsomes.[12] Although it has been said that the mortality from serum hepatitis is greater in heroin addicts, conclusive proof is lacking.

Tetanus, at least in New York City, recently has occurred mainly in heroin addicts. Subcutaneous injection ("skin popping") has been the most common route for infection with the clostridrium organism. The initial symptoms are usually stiffness or pain in the neck with trismus and chest pain, and the mortality has been nearly 90 percent in the reported cases. It should be noted that a patient recovering from tetanus is not completely immune to recurrent attacks and thus should be protected from further episodes of tetanus by appropriate re-immunizations.

The largest malaria outbreak in this country in 20 years occurred recently in Bakersfield, California. This outbreak was limited entirely to narcotics users. A total of 44 patients were affected. This disease was traced to one man, returned from Vietnam, who was using narcotics. Fortunately the strain of malaria in this outbreak was Plasmodium Vivax, which is rarely fatal. In the marshlands of the northern central valley of California there are still Anopheles mosquitoes which are capable of carrying malaria if they bite an infected individual. It is both interesting and disturbing to remember that the last large outbreak of malaria in California occurred in 1952 and involved 35 Campfire girls who were bitten by mosquitoes which presumably previously had bitten a returning Korean war veteran.

Bacterial endocarditis is another serious complication of narcotic addiction. To date 93 cases of endocarditis in narcotic addicts have been reported in the literature by 24 different authors.[13] In New York City over 8 percent of all deaths from narcotic addiction are the result of bacterial endocarditis.[14] The most commonly affected valves are the aortic and mitral valves and staphylococcus is the most frequently responsible organism. A history of underlying valvular heart disease is generally less frequent in addicts than in age-matched controls. Pulmonary manifestations of endocarditis are apparently very common. There is also an apparent increase in the frequency of tricuspid involvement, as well as infections due to Candida and enterococcus, in addicts although the literature is probably seriously biased toward over-reporting the unusual cases.

We have observed 10 to 20 cases of heroin-related bacterial endocarditis at the San Francisco General Hospital over the past year. Two patients had an unusual endocarditis due to Serratia marcescens. In this same regard, Nickerson recently reported striking increases in heat-stable opsonins of Escherichia coli and Serratia marcescens in heroin addicts.[15] A recent review suggested a very high incidence of endocarditis due to streptococcal viridans in heroin addicts.[13] This group also found that the endocarditis in heroin addicts primarily involved the left, not the right side, of the heart. However, when the lesions did occur on the right side of the heart the responsible organism was generally staphylococcus aureus. The mortality of endocarditis in heroin addicts is approximately 75 percent. Some postmortem findings in a fatal case, including a perforated aortic valve and ruptured cerebral mycotic aneurysm, are shown in Figure 2.

Since heroin in an unsterile vehicle is often injected under the skin, recurrent skin infections are frequent side effects of narcotic use. In a review of all heroin patients admitted to San Francisco General Hospital over the past year, I found that the vast majority were admitted for treatment of local infections or cellulitis due to unsterile injection procedures. Certainly cellulitis, thrombophlebitis and bacteremia are the major medical problems bringing heroin addicts to the San Francisco General Hospital.

Cherubin and Millian[16] have recently emphasized the nonspecific false positive reactions of many heroin addicts to a number of common serological tests. In their study of serological reactivity of narcotic addicts who were not in hospital

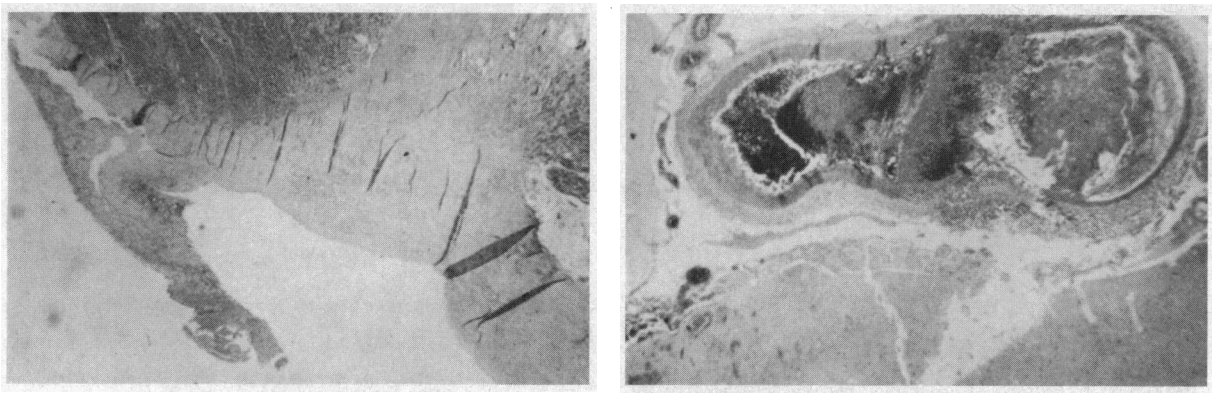

Figure 2.—Results of acute staphylococcus endocarditis in a heroin addict. *Left*, perforated aortic valve. *Right*, ruptured mycotic aneurysm.

and not in prison, they noted that nearly one quarter of them had a positive VDRL test. In those with positive VDRL tests, confirmatory tests for syphilis were positive in only 12 percent of the men and 15 percent of the women. They also found that nearly 20 percent of heroin addicts have a positive complement fixation test for lymphogranuloma venereum and 16 percent have a positive test for Q fever. Common serological tests therefore must be interpreted with caution in heroin addicts.

Sapira,[11] reporting from Lexington, Kentucky, emphasized a finding which we have observed repeatedly in the Detoxification Unit. Although we normally expect an elevated white blood cell count in patients withdrawing from alcohol or other drugs, we have found that the elevated leukocyte count associated with heroin withdrawal is predominantly due to an elevation in the absolute lymphocyte count, including some atypical lymphocytes and some Downey cells. Since many of the manifestations of heroin withdrawal resemble a flu-like illness—runny nose, abdominal discomfort and muscle aches—the syndrome of narcotic withdrawal may be exceedingly difficult to distinguish from a viral illness.

There are several other recently emphasized medical conditions which may occur in association with narcotic addiction. There is a striking increase in symptomatic hemorrhoids in heroin addicts, probably secondary to the constipating effects of long-term narcotic use. Several acute neurological problems have recently been observed. A crush syndrome after prolonged unconsciousness from narcotics with acute rhabdomyolysis and myoglobinuria, injection neuropathies and transverse myelitis have all occurred.

Citron[17,18] emphasized that a necrotizing angiitis which is indistinguishable from periarteritis nodosa may occur in a large number of young people with drug abuse. The patients upon whom he reported had used a multiplicity of drugs including narcotics, stimulants, hallucinogens and depressants. In some of these patients the duration of drug abuse was very short and the clinical spectrum varied from complete absence of symptoms in half the patients to multi-system manifestations including renal failure, hypotension, pulmonary edema, pancreatitis, gastrointestinal hemorrhage and hemolytic anemia. In addition to necrotizing angiitis, aneurysms of medium sized arterial vessels may occur in the kidney, stomach, liver, small intestines and selected viscera. Although the initial drug abuse in most of these patients was with amphetamines, many of the patients were using narcotics. Although the medical examiner in New York City reported finding no necrotizing angiitis in 1031 drug addict autopsies, the true incidence of this problem may escape pathological confirmation unless very careful autopsy studies are performed. Figure 3 shows diffuse periarteritis noted at postmortem examination of a 28-year-old heroin addict who had had repeated bouts of pancreatitis and died suddenly after (apparently) intravenous use of heroin in the hospital.

In conclusion, it is clear to those of us dealing with these patients that narcotic addiction is a serious and growing problem that will probably be increasing in magnitude rather than decreasing in the near future. Nevertheless, only about 15 percent of medical schools have in their curricula specific teaching programs concerning the management of drug abuse and alcoholism. The

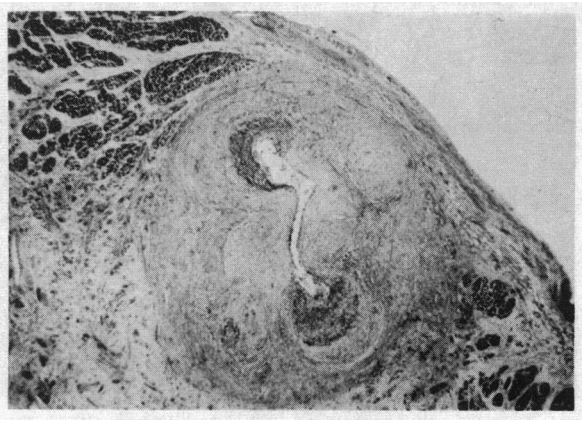

Figure 3.—Myocardial vessel illlustrating a striking clinically unrecognized periarteritis nodosa-like lesion.

old ways of dealing with narcotic addiction, including stringent laws and police enforcement, have been a failure from the point of view of rehabilitating the young drug offenders and correcting their illness. Imprisonment has done little but propagate addiction. As physicians, we can successfully treat the withdrawal symptoms and the development of tolerance in addiction, but we are still left with the primary behavioral problem for which little treatment and insight is curcently available. It is, however, clear that the pendulum of responsibility for the care of these patients is now swinging to the medical profession rather than the law enforcement agencies. We must therefore strive to find solutions to these problems.

TRADE AND GENERIC NAMES OF DRUGS

Amytal® ..... amobarbital
Nembutal® ..... pentobarbital
Seconal® ..... secobarbital
Nalline® ..... nalorphine
Lorfan® ..... levallorphan tartrate

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"SILENT" BLEEDING FROM INJECTION WOUNDS

A fair number of patients on heparin bleed because of trauma caused by *us*, . . . The most common problem has been giving intramuscular medications to these patients. Giving the medication with a large needle into the gluteal area has been a formidable hazard; several patients locally have bled to the point of requiring massive transfusion therapy. Unfortunately these patients often bleed into an area that's clinically silent (because the bleeding may be deep). It may not penetrate to the skin and cause surface ecchymoses. It may go down as far as below the knee and upward into the retroperitoneal area, and only after several days when the blood does surface to the skin is the clinician aware of this. One of the points to be made here is that if the patient is on heparin, it's wise to monitor the hematocrit every two or three days because this will be the first way of picking up unsuspected bleeding.

—PHILLIP S. WOLF, M.D., Denver

Extracted from *Audio-Digest General Practice*, Vol. 18, No. 11, in the Audio-Digest Foundation's subscription series of tape-recorded programs. For subscription information; 619 S. Westlake Ave., Los Angeles, Ca. 90057